THE AMERICAN JOURNAL OF

OPHTHALMOLOGY.

VOL. XII. FEBRUARY, 1895. No. 2.

ORIGINAL ARTICLES.

SIDEROSIS CONJUNCTIVÆ IN CONNECTION WITH A PECULIAR GROWTH OF THE OCULAR CONJUNCTIVA.

BY ADOLF ALT, M.D., ST. LOUIS.

E. T., colored, male, æt. 21, called on me April 25, 1894, on account of a tumor of the left eye. This eye had been injured and had atrophied some four years previous to his visit. About nine months ago the eye had, further, been greatly irritated by a cinder. (He being a railroad hand, this statement goes for nothing, as with them every eye trouble begins with "a hot or cold cinder"). From about that day on he noticed a swelling in the lower half of the eyeball, which grew steadily up to the time of his visit. He was employed as a car cleaner.

I found a phthisical eyeball. The cornea which was quite transparent, showed an indrawn scar passing in a horizontal direction from the center of the cornea into the ciliary region. The semilunar fold was greatly enlarged and from the lower fornix a bag-like swelling protruded which extended from can-

thus to canthus. Its surface was lobulated, its sides were smooth and depressed from the pressure of the lids, when closed. The tumor showed several millimetres outside of the palpebral fissure. It had a broad base. Its surface which, thus, was always exposed to air was pigmented in parts, of a brownish red color, very similar to the general pigmentation of the skin of the patient. There was no sign of inflammation at the time and hardly any discharge.

I removed the whole growth on the next day and found that it offered a considerable resistance to the scissors. A profuse hæmorrhage followed the removal and was controlled with some difficulty. Otherwise, the healing was in no way interrupted.

The tumor, when hardened in alcohol, was an inch long, one-fifth of an inch broad and a quarter of an inch high.

On sections the base and, in fact, the bulk of the growth is seen to be made up of very tough, dense connective tissue, resembling a fibroma, yet, I think, more likely due to hypertrophy from a chronic inflammatory process. In this tissue are embodied numerous large bloodvessels and between them a smaller number of spaces filled with blood-corpuscles; since these spaces, have no blood-vessel-wall, this portion of the growth must be looked upon as a beginning angioma cavernosum. Farther on the tissue of the growth shows patches of round-cell infiltration, which in their arrangement are very suggestive of tuberculosis. They, however, show no giant-cells and, moreover, the repeated search for the tubercle bacillus has been altogether unsuccessful. These round-cell patches lie in the deeper parts of the tissue and nearer the surface; they, however, in no manner resemble, as might perhaps have been suspected, the granules of trachoma. This suspicion was the more natural from certain changes in the epithelial cells which will be referred to a little later on.

In general, the epithelium covering the whole growth is very hyperplastic. Instead of the healthy conjunctival epithelium, there are many layers of cells, which are flat and horny on the surface and get more and more columnar and cylindrical to

wards the underlying tissue. The epithelium, furthermore, forms innumerable pegs reaching far down in the growth, which are so closely packed together that there is no room for any tissue between them, so that the tumor greatly resembles an epithelioma. It is lacking however all other essential particulars.

The peculiar pigmentation seen on the surface of the growth, which appeared like the pigment in the patient's skin, is seen to be due, instead, to particles of dirt, iron and coal dust, which lie in the cementing substance between the cells. Whereever these particles are located, they have followed the epithelium way down into the tissue as far as the epithelial pegs dip in, and lie usually in their peripheral parts, yet, they seem nowhere to have entered the connective tissue underneath. The occupation of the patient, as a car cleaner, is undoubtedly responsible for this particular siderosis.

There are some further peculiarities found in the epithelial layer. A considerable number of glands penetrate from it into the depth to a varying degree. These glands are of the acinous type, opening on the surface with a wide duct. They are lined with an epithelium the cells of which are large roundish and oval, vesicle-like structures, here and there somewhat flattened against each other. Their nucleus always lies to one side near the periphery of the cell.

Whether these glands are newly formed in the tumor or whether they are simply Krause's glands which during the growth of the tumor have been drawn out of their position in the fornix conjunctivæ onto the surface of the new-formation, I am unable to decide, but I think the latter very probable.

However, these same oval or round vesicle-like cells are also found in great numbers and in many places in the epithelial pegs outside of these glands. Here they lie almost uniformly in the middle layers of the thick epithelial coat. Their longest diameter is not parallel with that of the other cells and they seem to reach to the surface in but few instances. Hæmatoxyline, eosine, carmine and methyl-blue do not stain the body of these cells. They have, however, a strange affinity for Bismark brown. This coloring substance they take up to a much higher degree than the surrounding

cells and tissues, so that they appear a dark black-brown.

I have seen, what appeared to be the same structures, many a time before in the conjunctival epithelium and have always looked upon them as cells undergoing a mucous degeneration which would later on be goblet-cells. Never, however, have I seen them in such immense numbers.

They appear exactly like the structures found and described by N. C. Ridley ("Some Points on the Histology of Trachoma," Transactions of the Ophthalmological Society of of the United Kingdom, Vol. XIV., p. 24, etc.), and depicted on Pl. I., which he considers to be probably protozoa (perhaps gregarinidæ).

He gives the following reasons for concluding that these structures are not future goblet-cells:

1. They are far too numerous.
2. Some are found almost alone at a part where goblet-cells are not ordinarily found, and sometimes they are present in squamous epithelium.
3. Many are not in contact with the free edge of the epithelium as goblet-cells should be, but are in the deepest layers.
4. Many have their long axis not at right angles to the surface, as is the case with the goblet-cells, but in various directions, and in some cases even parallel with the surface.
5. Their reaction to staining fluids differs.

These points, thus mentioned by Ridley, almost exactly correspond with what is seen in the epithelium of the growth under consideration. Only in the glands the condition differs in so far, as almost all the epithalial cells within them appear of the same nature. If these structures are protozoa, this would not be so very astonishing, as the glandular cavities would serve very well for an appropriate domicile.

Ridley does not state, whether he has stained them with Bismark-brown. Certainly their especial affinity to this aniline-color is in my specimens so remarkable, that it is worth while to examine further into it. I know of no particular cells in the human organism which show this affinity. I am, therefore, the more inclined to accept Ridley's views on this subject.

TRANSLATION.

PARESIS OF THE ABDUCENS NERVE AND PNEUMONIA.

BY C. H. A. WESTHOFF, M.D.

November 16, a woman brought her little daughter Kath., two and a half years old, to my clinic.

The child was led by the mother's hand, the head turned to the right. While she stood before me, the head continually remained turned to the right, when I held her head firmly and placed the mother to the right of the child and ordered her to call to the patient, I saw, that the right eye could not be moved outwards. It remained fixed in the median line, while the left eye was moved fully towards the inner canthus. The movements of the right eye were normal in an upward, downward and inward direction.

There existed, then, a total paralysis of the right abducens nerve. The turning of the head towards the right, was done to escape the double vision. The ophthalmoscope showed nothing abnormal.

My colleague, Dr. Voûte, specialist for children's diseases, who held his clinic at the same time, was kind enough to examine the child and to report to me the following facts:

The child is the seventh one of a father 37 years old, and a mother, 35 years of age, who are both healthy. Having been fed artificially, it shows some degree of rachitis. No trace of lues. The child who formerly was always well, was suddenly taken with a vehement fever about two weeks previ-

ously. After the fever had lasted three days, the mother noticed the peculiar position of the head and had therefore consulted an oculist. The physical examination demonstrated a considerable apex pneumonia of the left lung. The cough was sparse; hardly any fever; all other organs were normal.

A week later the pneumonia was considerably improved. The motility outwards of the eye had increased. Another five days later the improvement was considerable and *pari passu* the paralysis of the abducens had grown much less noticeable.

December 7, but a very slight dullness could be found upwards and backwards and the paralysis was all but cured.

A few days later everything was normal. In this case, then, we had a paralysis of the abducens nerve which developed a few days after a considerable apex pneumonia had come on. At the time when then the pneumonia was at its highest, the paralysis was complete; and as the pneumonia disappeared, the paralysis grew less, and disappeared totally when the lung had regained its health.

It is, therefore, probable that we must assume a common cause for both affections, especially since no other cause, like lues, could be found.

In the literature at my disposal I cannot find anything concerning such a combination. Oppenheim alone says, without going into details, that paralysis of the oculomotor nerve has been observed after pneumonia.

It seems to me that from analogy we may assume a connection between the two affections. We often see paralyses of the accommodation after diphtheria. Donders was the first to prove the connection between these two affections. It is generally assumed that the paralysis is due to an intoxication of the nerve which governs the accommodation, by the toxines, which are produced by the bacillus of diphtheria.

We do not know, how this intoxication takes place, and why after diphtheria usually the same nerves are paralyzed; and we can as yet give no explanation.

The fact, however, that in diseases in which, after an in-

fection with bacilli, toxines are taken up by the blood, paralysis may develop, gives us a right in our case to assume a common cause for the pneumonia and the paralysis of the abducens, and this the more, since the healing of the two affections progressed *pari passu*. As soon as, due to the decrease of the focus of infection, less toxines entered the blood, the paralysis, too, grew less and when no more toxines were formed, it disappeared.

It will, therefore, in the future be well in all cases of ocular paralysis to know whether they may not be due to an infective process and the consequent formation of toxines which may enter the blood.—*Centralblt. fuer prakt. Augenheilk.*

TATOOING OF THE CORNEA IN ORDER TO IMPROVE THE VISUAL ACUITY.

BY OTTO LANDAU, M.D.

On October 2, 1894, W., aged 28, came to consult me and to see whether the condition of his right eye, which about two months previously had suffered from a vehement and prolonged inflammation, could not be improved. I found the conjunctiva free from any irritation and a large, almost non-translucent, leucoma of the cornea, which was of a rectangular shape and with its irregular and semitransparent edges amply covered the lower three-quarters of the pupil. With its lateral margins it reached the limbus corneæ. At its temporal edge the iris was adherent in one point. The visual acuity was barely $^1/_{50}$ for distance, with + 6 D. Sn. $3^1/_2$ at 4 inches; the visual field and tension were normal. I proposed to tatoo the leucoma and to perform an iridectomy later on, in order to improve the looks and to improve vision by means of the blackening of the leucoma.

On October 9, the leucoma was tatooed in its whole extent in one sitting, the needles being introduced obliquely. Of course the anterior synechia was not interfered with. The patient held so still, that no fixation forceps was needed. After the operation the leucoma was deeply black. Four days later the cornea had healed without irritation, was black, smooth and reflecting well.

On October 30, V. for distance was $^{15}/_{LXX}$ without glasses, that is almost $^{1}/_{5}$ and Sn. $1^{1}/_{2}$ was easily read at 6 or 7 inches.

This considerable improvement of the visual acuity by the tatooing of the leucoma alone, made me abstain from an iridectomy.

The case is closely allied to the one published in the *Deutsche Medicinische Wochenschrift* by my revered teacher and former chief, Prof. Hirschberg and proves that a better visual acuity is obtained by tatooing alone, without iridectomy, if but a very small space of the natural pupil is free.

Furthermore, it seems that the oblique introduction of the the needles into the leucoma (which can be done splendidly in one sitting, since the whole field can be overlooked and even the semitransparent edges can be well blackened) is preferable to the methods of Baiardi and Liebrecht, in which a needle is entered under the surface of the cornea and the ink at the same time with it or injected afterwards by means of a Pravatz syringe.—*Centralbl. fuer prakt. Augenhlk.*

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.

Section of Ophthalmology and Otology.

General Meeting, October 18, 1894. D. B. St. John Roosa, M.D., President.

Discussion on Defective Vision in Its Relation to Crime.

Dr. Frank Van Fleet read the paper with this title. See December, 1894, number of this journal.

Dr. Henry Dwight Chapin.—I have been intensely interested in this paper, which has suggested many lines of thought. The general subject of increase of crime is one of much importance. Some years ago I looked up the subject of increase of insanity and pauperism, and published my results in an article on "The Survival of the Unfit." It was shown there that the criminals, paupers, and insane were increasing out of proportion to the population. The relative importance of heredity and environment has been treated in a novel manner in this paper. I am glad to hear the argument advanced by the author that environment is so much more important than heredity. Heredity is out of our reach, but environment is more or less under our control, so that it is, at any rate, a pleasant doctrine. I am inclined to believe that the influence of heredity has been, at times, over-estimated. My thoughts were first turned in this direction some twelve years ago, when I was put in charge of an institution known as St. Christopher's Home. It had twenty-five little girls, vary-

ing in age from two to ten years. These children had been taken from the poorest tenements, and their parents had come from the worst classes. I devoted considerable time to studying their development and surroundings. For several years there was much sickness in the institution—cutaneous diseases and catarrhal affections. After a while, under improved environment, these affections entirely disappeared. I believe that a vast deal can be accomplished, physically, by securing favorable environment for little children. I am not sure that so much can be done morally. It is not an uncommon experience to find children whose physical well-being has been carefully looked after, nevertheless remain immoral.

Now, in regard to ophthalmia neonatorum—what are we to do with the many cases of this kind seen in dispensary practice? The mother is utterly incapable of looking after such eyes, yet I know of no eye hospital that will take charge of these cases. If we are to save these children from blindness, as we all know we can do by proper treatment, we must make better provision for these poor little ones than exists at present. Some years ago I inspected about twenty public schools, and I found that the primary school children—those who remained the shortest time in school—had the poorest accommodation. The lighting and ventilation of these schoolrooms were very bad. Many of them were lighted by artificial light, and, in addition to this, there were many conflicting lights. If what we have been told about defective vision producing criminals is true, we are likely to have a pretty large criminal class in New York City.

DR. JOSEPH COLLINS.—I differ from the author on very many points. It seems to me that the origin of crime can hardly be satisfactorily divided into physical and moral, for this leaves out entirely the cosmic element, it partly excludes the biological element, and it includes only the social element. It has been proved by students of criminology that the cosmic influences of crime are not to be underestimated. During the winter, crimes against property are common; on the other hand, during the spring and summer, crimes against persons

are common. The reader of the paper has said that education has been asserted to increase crime. Yes, that is, intellectual education, but the education which decreases crime, and which has not been spoken of, is manual education. This is the consensus of opinion of all practical anthropologists; hence, in speaking of education, we should not overlook physical education. If children having criminal antecedents are taught trades and the means of earning their livelihood thus given to them, it is probable that we shall in this way decrease crime, even though they may be slightly handicapped by some slight somatic deficiency. If, however, we attempt to educate their warped intellects we shall probably give them just enough acumen to encourage them to commit crime. It is strange to listen to a paper on crime in which the element of heredity is relegated to the background, without hearing of any of the views brought forward by August Weissmann. It is novel, likewise, to hear reiterated the views advocated many years ago that environment outranked heredity. I think we should not confound acquired characteristics with the phenomena of heredity. Although it is a fact that such procedures as circumcision are followed by the birth of children with foreskins, it is necessary to keep in mind that acquired characteristics are not hereditary, and, carrying out this argument further, we can show that no acquired characteristics are handed down to posterity. Heredity, however, is something much more obscure than this, and to consider it all, it must be approached from the biological standpoint. If crime is due more to environment than to heredity, and we can improve environment, how is it that crime is still on the increase? If, as Superintendent Byrnes says, crime is due to drink, as it is frequently, then I should like to ask if inebriety is not an evidence of heredity. As regards criminals begetting criminals, Superintendent Brynes has reported a number of instances where criminals have endeavored to place their children in the best environment and give them the best education, and yet some of them have become criminals like themselves. The bastard son of Napoleon, notwithstanding the best of environment, was

frequently a fugitive from justice. Why has it become a notorious fact that the sons of the most noble profession very oftem stray from the path of rectitude?

I would like to ask if the statistics of criminals show that most of them have bad eyes. The instinctive criminal has defective eyes, but has also defective smell, defective taste, and defective sensory conduction. I wish to emphasize the fact that a paper on such a subject should not dispose so hurriedly, and without a proper consideration, of such important factors as heredity and environment.

DR. FREDERICK PETERSON.—I was rather surprised to find so much stress laid on defective vision as a cause of crime. I have been under the impression that most people have defective vision; and, furthermore, although the Arabs and Egyptians are almost uniformly affected with some ocular defect, crime among them is exceedingly rare.

The author has put forward some very radical ideas regarding heredity. I am afraid that Weissmann would be compelled to give up his studies if the views just presented are adopted. It seems to me that there can be no doubt that heredity is infinitely more powerful than environment.

DR. J. E. WEEKS.—Defective vision affects two-thirds of all the human race. In ophthalmic practice, it is quite rare to find a perfect eye. A few days ago I examined the eyes of a young lady, and found them perfect as far as optic principles were concerned; this is the only case I have seen in my office for three or four years where both eyes were optically perfect. It is not surprising that there should be such a percentage of defective vision in inmates of institutions as that mentioned by the writer of the paper.

The mere testing of the ability of more or less illiterate children to read certain type on a card is certainly a very superficial method of examining the vision, and should not be employed to draw important conclusions from. Notwithstanding our increased knowledge of the methods of correcting defective vision, it is stated that crime is on the increase. This should not be if defective vision is an important factor in caus-

ing crime. It has possibly something to do with the production of crime, just as defects of the other senses are related to the production of crime, but, in my opinion, to no greater degree.

If cases of ophthalmia neonatorum were seen at an eye dispensary every day, there would be a very small percentage of blindness from this cause. I do not think that more than two or three per cent of the cases of ophthalmia neonatorum which have been treated as out-patients at the New York Eye and Ear Infirmary have suffered impairment of vision. It is, however, necessary that the children should be seen every day. Under these circumstances, it does not seem necessary to have a special hospital provided especially for such cases.

DR. ROOSA, the President.—I would like to say a few words on the subject of contagious ophthalmia in addition to what has been said by the last speaker. The responsibility for the present condition of affairs should be placed where it belongs—on the officials of the city, for they have refused to erect a special hospital for the care of contagious eye disease. The Manhatten Eye and Ear Infirmary Hospital and the Eye and Ear Infirmary receive all the cases they can possible take care of, but usually daily attendance is sufficient for ophthalmia neonatorum, and the eyes are saved if there is any one at home to carry out the directions. If we received in the hospital all the contagious cases that apply, it would be necessary to stop taking care of cataract, glaucoma, and all operative cases. It is certainly outrageous—for I will use as strong a term as that—that there is no special hospital in the city for contagious eye disease.

DR. VAN FLEET.—I can only repeat what I have said that heredity must sink into insignificance alongside of environment if we look the subject squarely in the face. It seems to me that the opinion of a practical man like Superintendent Byrnes is worth more than that of a theorist, no matter how prominent he may be in the scientific world.—*American Medico-Surgica Bulletin.*

SELECTIONS.

CORTICAL LESION OF THE LEG, SHOULDER AND VISUAL CENTRES OF RIGHT HEMISPHERE.

BY L. R. CULBERTSON, M.D., ZANESVILLE, OHIO.

The subjoined case is one of unusual interest, because of the peculiarity of the paralysis of chiefly the extensors of the leg and arm, and of the visual centre of one eye.

Master J. T. N., aged thirteen years. V., R. E. Rem. unaided=1/3 Sn. Ophthalmoscopy shows he requires +.5 D. sp. each eye. Has marked left internal strabismus. Pupils do not react to accommodation, but they do to light, showing cortical lesion. Irides dilate normally to atropine; no synechiæ. Corneæ normal.

Ophthalmoscopic examination: R. E. vitreous, lens and choroid normal. Disc very pale, and shows atrophic cupping. Both arteries and veins too fine; capillaries wanting on disc. Has had strabismus of L. E. since birth, due to paralysis of left sixth nerve. Eyes or body not affected by measles, scarlet fever or whooping-cough.

His father says he never had syphilis, and there is no history of syphilis on either side of the house. Formation of teeth normal, and no enlarged glands, etc. No tuberculosis in family. His father says forceps were not used, and he was not injured about the head at birth. Also has always been defective in strength on the left side. Cannot supinate left hand; cannot touch back of head with fingers of left hand; cannot touch centre of back with left fingers (paralysis of infra-spin-

atus); deltoid normal. Right biceps measures ten and one-half inches, left nine and one half inches. Infra-spinatus wasted. Right forearm measures nine and one-half inches, left eight and one-half inches. Cannot button coat with left hand; cannot stand on left foot with eyes closed. Left knee-jerk slightly exaggerated, right normal. Cannot flex left foot (posterior muscles affected), and cannot flex toes of this foot; has only partial extension of left foot; extension of left toes normal. Calf, right leg fifteen inches, left fourteen inches.

Esthesiometry: Tip of tongue normal; palmar surface left distal phalanx three lines, right normal; face normal, both sides; great toes normal. All other points normal.

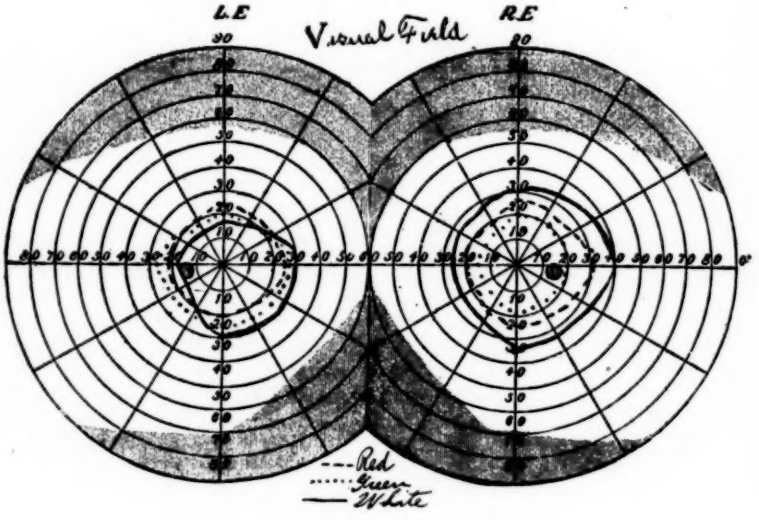
The right side of the head is much smaller than the left. There is very marked difference in development between the two sides of the cranium in the temporal and fronto-parietal regions. Measurements show marked difference in the two sides of the cranium. Boy seems somewhat defective in intellect. When told to do anything he seems to have some difficulty of comprehension. Hearing normal in both ears. Walks in straight line with eyes closed. Writes well with right hand, but cannot write with left (is right-handed). No agraphia, aphasia, amnesia nor alexia. Color vision normal. Pain sense normal; heat sense normal; no nystagmus. Both parents have well-shaped heads. Urine 1020, no albumen, no sugar. Gave strychnia treatment. He never had convulsions or epilepsy.

REMARKS.

We have here a difficult task to diagnose the cause of the lesion of the cortical centres. First, let us investigate the left-sided paralysis. The *essential paralysis* (*anterior poliomyelitis*) of children comes in infancy, usually during dentition,[1] and the fundus of the eye shows no diagnostic symptom, so far as I can ascertain from ophthalmic literature. Anterior poliomyelitis does not occur before or soon after birth. Two diseases that might cause these symptoms are *chronic hydrocephalus* and

[1]Da Costa's "Diagnosis," p. 120.

hypertrophy of the brain. In chronic hydrocephalus, as the disease progresses the head becomes very large, the gait tottering and uncertain. As the malady advances, strabismus, partial palsies, epileptic convulsions, vomiting, loss of sight, anæsthesia, etc., are observed.[2] Dr. J. S. Ramskill[3] says that



in some cases of chronic hydrocephalus the openings between the ventricles become closed, causing the fluids to accumulate more in one part than in another, thus causing unsymmetrical development of the head. My patient does not present the appearance of a hydrocephalic lesion and none of the symptoms save the one-sided enlargement and paralyses. In *hypertrophy of the brain* we find severe headache, stupor, convulsions, palsies, according to the centres pressed upon, and optic neuritis.[4] My case is lacking in most of these symptoms. His cranium is certainly rachitic, and there may be thickening of the inner table, right side, over the cortical areas of the paral-

[2]Ibid.

[3]Reynold's "System of Medicine," p. 837.

[4]Da Costa.

yzed muscles; yet if it were due to pressure we would expect that at some period of his life he would have had epileptiform convulsions or epilepsy, but he has not. We exclude tumor of the cortex by absence of history of epileptiform seizures and epilepsy. Very careful inquiry fails to elicit any history of a fall or blow on the head.

Summing up all the symptoms and history of the case, the most plausible opinion is that there has been a thickening of the inner table over the cortical area affected, and the absence of epilepsy and epileptiform seizures may be accounted for by the fact that the right half of the cranium is so abnormally large at other points that it, to a certain extent, compensates the pressure over the affected area.

The cortical centres involved, according to Gowers, are the posterior part of the superior frontal convolution, the upper parts of the ascending frontal and ascending parietal convolutions, the upper and anterior part of the superior parietal lobule, and a part of the mesial surface of the brain (all of these the centre of the lower limb); the upper limb centre, *i. e.*, the middle third of ascending frontal and ascending parietal, and the shoulder centre lying in the upper part of this area. Referring to Ferrier's work,[5] we will see, according to his plate, that centres 2, 3, 4—used in climbing and walking—are affected. These govern some of the muscles of the arm and leg, being situated about the fissure of Rolando. Also parts of the posterior central convolution, *a*, *b*, *c*, *d*, governing movements of the hand and wrist, as in clinching the fist.

We cannot definitely locate the lesion to the sixth nerve. H. R. Swanzy[6] says "there is no nerve so liable to provide a distant symptom." Gowers refers this liability to the lengthened course these nerves take over the most prominent part of the pons, which renders them readily affected by distant pressure. Prevost[7] says that "in paralysis of the muscles govern-

[5] "Functions of the Brain," p. 305.

[6] "Bowman Lecture" in British Medical Journal.

[7] Ibid.

ing conjugate deviation the affected eye always looks toward the side of the cerebral lesion." This is true in my case.

In locating the optic lesion, Wernicke's pupil symptom confirms diagnosis of primary cortical lesion. The fact that neither pupil reacts to accommodation, but does to light, might be explained in the same manner as in the case described by Berger and reported by Wernicke,[8] in which there was blindness of both eyes and both pupils reacted to light. Autopsy showed cortical lesion of one side and disease of optic tract of the opposite side.

So many years have elapsed since the onset of the disease of the eyes that it would be impossible to decide whether the atrophy of the optic nerves is due to pressure on cortical centres, causing a descending optic atrophy, with crossed hemianopsia, and associated with descending neuritis of right optic tract, and later the right and left optic nerves (there is slight hazinesss about both discs); or whether there has been not only pressure on the cortical visual centres, but also on the right optic tract, causing neuritis of both optic nerves.

It is probable that the primary lesion was cortical, being situated in the inferior parietal lobule, including the supramarginal convolution and angular gyrus with the subjacent white substance, which can cause hemianopsia with or without other symptoms.[9] These centres are contiguous to the motor areas affected, and may have been pressed upon or affected by a localized meningitis. This may have been a case of crossed hemianopsia in the beginning, but optic neuritis has changed its hemianopic character. Abercrombie[10] reports a case where injury to vertex caused paralysis of right hand, right leg weak; later clonic spasms of facial muscles, arm and leg, hemianæsthesia, right lateral hemiopia, optic neuritis. Autopsy showed a round-celled sarcoma occupying the (left) occipital and part of the parietal lobe, convolutions of anterior

[8]Loc cit., Bd. iii, p. 335.

[9]Seguin—Archives de Neurology, Vol. xi, 1886, p. 176.

[10]Transactions of the Ophthalmological Society of the United Kingdom, 1887.

half of occipital lobe, superior parietal lobule and upper extremity of angular gyrus thinned. The optic neuritis was ascribed to chronic thickening of the meninges at the base of the brain. The concentric contraction of the visual fields was probably due to optic neuritis.

According to Ferrier,[11] unilateral destruction of the angular gyrus causes temporary abolition or impairment of vision in the opposite eye not of a hemianopic character. Dr. Gowers[12] says, "numerous observations have established beyond question the fact that hemianopsia results from disease of the occipital lobe, which thus constitutes a centre for the fibres from the same-named half of each retina, and receives impressions from the opposite half of each field of vision. Complete hemianopsia has most frequently been produced by disease of the apex of the lobe, and especially of the cuneus." He believes, however, that, in addition to this half-field visual centre, there is in front of the occipital lobe a higher visual centre, in which the half-fields are combined and the whole opposite field is represented. Such a centre he believes to be localized in the angular gyrus, basing his belief, first, on the cases of "crossed amblyopia," where post-mortem evidence has shown this region to be affected, and also on the experiments recorded by Ferrier. Swanzy[13] says that, in crossed amblyopia—in which the side away from the lesion is almost blind, with very contracted field, while the field of the other eye is also contracted, but in a less degree—the lesion has been found in the lower and hinder part of the inferior parietal lobule.

"Pathological anatomy leaves no doubt that, in man, the visual centre is situated in the occipital lobe rather than in the angular gyrus and elsewhere, and the evidence goes to show that the absolute optical centre chiefly occupies the cortex of the cuneus, and of the superior occipital convolution, and also especially in respect to the color sense, the posterior part of

[11] See M. A. Starr's article in Am. Jour. of Med. Sciences, 1884.

[12] "A Manual of Diseases of the Nervous System."

[13] "Bowman's Lecture," in Transactions of the Ophthalmological Society of the United Ringdom, 1888.

the superior and inferior occipito temporal convolutions."[14] Color vision was not affected in my case.

Ferrier[15] says that bilateral destruction of the angular gyrus and occipital lobe together produces complete and permanent blindness, followed by atrophy of the optic disc and fixity of the pupils, all other sensory faculties and motor powers remaining absolutely unimpaired from first to last. Destruction of the angular gyrus and occipital lobe of the same side produces temporary blindness in the opposite eye, and more or less enduring hemianopsia in both eyes towards the side opposite the lesion. The restoration of vision in this last case Ferrier believes due to incomplete destruction of the centre. This it is extremely difficult to avoid. "After total extirpation of both angular gyri it is doubtful whether complete clearness of vision is ever regained. The occipital lobes, however, can be injured or cut off bodily, almost up to the parieto occipital fissure, on one or both sides simultaneously, without the slightest appreciable impairment of vision-"[16] This has been repeatedly verified by Ferrier and Yeo, and confirmed apparently by Horsley and Shäffer, it must for the present at least be accepted. Ferrier amends Charcot's well-known diagram of the optic tracts and visual centres, giving each occipital lobe what we may call a hemiopic relation to the retinæ, this latter partly by fibres crossing in the chiasma, partly by fibres crossing in the lower visual centres, possibly the corpora quadrigemina."

Haab[17] records two cases, "one observed by Prof. Huguenin, in which homonymous hemianopsia was proved to be due to a lesion situated in the median surface of the right occipital lobe of the cerebrum, close to the posterior limit. Huguenin's case was a girl, aged eight years. Symptoms were dullness of

[14]Haab—Monatsbl. f. Augenheilk., 1882, p. 149. Huguenin—Monatsbl. f. Augenheilk., 1882, p. 143. Seguin—Arch. de Neur l., 1886, p. 176.

[15]Ferrier—"The Functions of the Brain," Second Edition, 1886.

[16]Ferrier—Second Edition.

[17]Haab—Klin. Monatsbl. f. Augenheilkunde, 1882.

intellect, headache, and irritability; enlargement of the head; vomiting, and later, optic neuritis; convulsions, beginning in the facial muscles of both sides, passing to the arms and legs, and followed by unconsciousness for half an hour or so; hemianopsia (right retinæ); no deviation of the eyes, no ptosis, pupils equal and active, no loss of sensation. Diagnosis during life, hydrocephalus of unknown cause; after death a caseous tubercular tumor was found at the apex of the left frontal lobe, occupying the bend of the second frontal convolution, and measuring 2.5 cm. long, 1.76 cm. wide; the second seated directly in the right sulcus hippocampi, in the median surface of the posterior part of the occipital lobe of the cerebrum, and measuring 3 cm. high and 2.5 cm. thick."

Lastly, in closing this article, I will say that the sutures of the skull on the left side may have become prematurely ossified, thereby pressing on the brain, but this theory could hardly hold good, as the symptoms show right-sided cortical lesion.—*Cincinnati Lancet-Clinic.*

A CASE OF HÆMORRHAGIC GLAUCOMA—UNUSUAL HISTORY—A SUGGESTION AS TO WHY SOME CASES OF RETINAL HÆMORRHAGE ARE FOLLOWED BY GLAUCOMA AND OTHERS ARE NOT.

BY JOHN DUNN, M.A., M.D., RICHMOND, VA.,

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Within the past few years, I have had under my care three cases of hæmorrhagic glaucoma.

In the *first case*, Mrs. P., aged 68, I removed the eyeball as a primary procedure; the glaucoma was at the time absolute,

and the inner parts of the ball were found to have undergone many inflammatory changes.

In the *second case*, Miss P., aged 65, I attempted an iridectomy which resulted in a dislocation, from sudden relief of tension, of the lens into the anterior chamber; the corneal wound was on the second day enlarged, and the lens allowed to escape; with it came the entire contents of the ball, followed by a profuse hæmorrhage, which did not stop until enucleation was done.

The *third case*, I was fortunate enough to see in the preglaucomatous stage, and to have under my immediate care during the progress of the disease; and as it offered one or two suggestions as to the manner of development of one form of hæmorrhagic glaucoma, I have thought it worth the while to report it.

Mr. P., aged 65. Syphilis in early manhood. A steady drinker for years. Full habit. On February 24, 1894, patient retired to bed in good health. On arising the next morning, he noticed that the sight of his left eye was practically gone. He could distinguish, however, light from darkness. No pain about the eye. No external inflammatory signs. On March 6, he consulted me. Tension of ball normal. Examination showed the fundus of the eye to be the seat of innumerable hæmorrhagic areas of greater or lesser size. They were most numerous and largest about the macula. The region of the optic disc was much swollen; vessels of disc, save for short sections of them, concealed; many hæmorrhages about the swollen disc. The hæmorrhages were less numerous in the outer than central parts of the fundus. Vision, perception of light, and of outlines of objects held peripherally.

O. D. $V=^{18}/_{XVI}$. Fundus is interesting. It is the seat of very numerous deposits, which, although occurring in greatest numbers in the macular region, and in the region just adjacent to the disc, are scattered throughout the fundus. These deposits, more or less circular in form, vary much in size; in places they resemble drops of rain, in other places they are pearly in color; there is no pigment deposit about them; they

occur alone, or in groups covering larger or smaller areas; they cannot be said to follow the lines of the retinal vessels; for, while, in some places, they seem to be ranged on either side of a vessel, they are just as numerous and as large in regions where the retinal vessels cannot be seen with the ophthalmoscope. (Urine normal; heart normal; lungs normal). The optic disc itself presents nothing that would attract attention in an otherwise healthy eye. Perhaps the veins are a trifle dilated, and the arteries somewhat contracted. They are not enough so to attract attention. The seat of these deposits must be the choroid, for a retina with such changes would necessarily show a corresponding diminution in visual acuity. V., in the case of Mr. H. is $^{18}/_{XVI}$, and accurate for both far and near vision.

Mr. H. was given the iodide of potash internally. Hot baths to the eye. Irritation to the skin of the temporal region. Under this treatment, the swollen condition of the disc diminished considerably and there was some absorption of the hæmorrhages.

On March 25, the eyeball began to pain and to be slightly injected about the corneal margin. Incipient glaucoma was suspected, although there was no increased tension. Treatment proved of no avail.

On March 31, the tension of the ball began to get above normal. Eserine (gr. j—℥j) was ordered to be put into the eye twice daily. It produced such pain that the patient (I was out of town) decided, after the second instillation, to await my return. (And I may say here that in this form of glaucoma eserine is contra-indicated).

On April 2, the eyeball had become slightly painful on pressure; while it was constantly so painful as to cause patient to desist from everything save attention to the eye.

On April 3, the cornea had begun to become cloudy; the fundus could not be made out by the direct method, while the appearance of two small hæmorrhagic spots on tbe surface of the iris showed, it would seem, that *the iris was diseased.* The

tension was, however, at this time very little above the normal. Pupil not more dilated than its fellow.

April 4. Pilocarpine, gr. $^1/_6$, twice daily; hot applications to the eyes. Tension normal. Disappearance of the hæmorrhages on surface of the iris. Eyes less painful.

April 5. Pupil irregularly dilated, the lower part of the circle being somewhat flattened, while thrown off from its edge is a small amount of pigment or blood—which, I cannot determine. Tension normal. Ball, however, much injected, and some tendency to œdema of bulbar conjunctiva. Pupil responds less actively to light than in other eye.

April 9. Pilocarpine has been continued. Tension above normal until April 8, when patient was bled from the temple. Conjunctiva remains injected, especially about the cornea. Fundus cannot be made out distinctly. Slight pain on pressure. Eyeball more or less continuously uncomfortable. No further iritic hæmorrhages. Hot applications.

April 16. The symptoms have varied in intensity since above date. General tendency, however, toward increase of the glaucoma. There has been increase in amount and extent of area of deposit along the lower border of the pupil. Marked increase of tension towards night, and with it increase of pericorneal injection. On April 12, put into eye several drops of 4 per cent. solution of cocaine for the purpose of examination of fundus. Pupil dilated irregularly; *dilatation caused appearance of one fairly large and several minute hæmorrhages from surface of iris.* Fundus cannot be made out because of deposit from iris edge on lens, and of some cloudiness of cornea, chiefly in lower half. Eye feels *sore*, and is tender of pressure. On April 14, patient was again bled from temple; tension immediately fell below normal. Anterior chamber deeper than its fellow. Pilocarpine and iodide of potash continued. Eye presents appearance of iritis rather than of glaucoma.

April 24. Condition of eye has changed but slightly. The pericorneal injection is a more firy red; the tension remains above normal; there have appeared on the surface of the iris numerous hæmorrhagic spots, very small, but distinct.

Pain in ball more or less constant. Some increase in amount of pigment deposit about pupillary margin.

July 15. Hot applications and use of potash continued. The redness has left the eye. The hæmorrhages in the fundus have disappeared, save minute ones here and there—evidently of recent date. The iris is still adherent to the lens along lower part of the pupil. Tension of the ball is slightly plus. There is no pain. Vision has slightly improved. No swelling about nerve.

November 15, 1894. Patient has suffered no inconvenience in O. S. since last examination—in July. Vision has slightly improved—the eye is, however, still useless for the purposes of accurate vision. No hæmorrhages can be made out in the fundus. Pupil attached below at margin to the lens. Tension of eyeball decidedly suspicious.

The above case has several interesting points. We have, as the starting-point of our trouble, choroidal changes present in both eyes. These changes were, in all likelihood, vascular, and resulted in rupture of the vessel walls. One month later, signs of iritic irritation showed themselves, and, with them, signs of glaucoma. A few days later, the high tension was well marked, and the iritic irritation sufficient in degree to cause adhesions between the iris edge and the lens. Slight hæmorrhagic extravasations appeared on the surfaee of the iris, while even such a mild mydriatic as a weak solution of cocaine caused, in the act of dilatation of the pupil, further iritic hæmorrhages. I think there was at no time a true inflammation of the iris. The interpretation of this sequence of events is, it would seem, that numerous particles of the diseased tissues of the blood-vessels separated, at the time of the hæmorrhages, into the fundus, were carried by the lymph current into the filtration angle, where they lodged, causing obstruction to the lymph outflow—hence the high tension; some of the diseased particles were also carried into the iris tissue—hence its signs of irritation. I think we have, in this case, an indication as to why some cases of retinal hæmorrhage are followed by hæmorrhagic glaucoma, and others are not. Where

the extravasation into the fundus is purely of the blood-elements, there is no likelihood that the absorption of this extravasation will lead to glaucomatous symptoms; where, however, the extravasation consists not only of blood, but of a considerable number of particles of diseased choroidal tissue—in reality, foreign bodies—their removal by the lymph current may produce glaucoma. These particles of diseased tissue reach the filtration angle as emboli, lodge in the minute channels of escape for the fluid from the inner parts of eye, act as irritants to the walls of the lymph channels in which they are lodged, set up an inflammation varying in degree according to the number and character of the emboli and their size, produce, as a result of their irritative action, occlusion of the lymph channels in which they are lodged, and thus arise the symptoms of glaucoma.

The changes noticed in the choroid in the above case may have been amyloid in nature, and the hæmorrhages may have detached numerous minute particles of these diseased areas. Once detached, these particles of diseased tissues would serve as foreign bodies, and then the sequence above noted. I do not mean to say that this is the invariable cause and sequence in the hæmorrhagic glaucoma. The inference from this one case is that hæmorrhagic glaucoma may have such a cause.

The question may be asked: Will this eye finally be lost through glaucoma, so that enucleation will be necessary? At the present time, eight months have passed since the first glaucomatous symptoms were noticed. Mr. H. still retains the eye, which is the source of no inconvenience to him, save such as arises from imperfect sight. Should fresh hæmorrhages occur, it is very likely that further glaucomatous symptoms will appear, and repeated hæmorrhages, accompanied by separation of particles of diseased choroidal tissue, would certainly be followed by fully developed hæmorrhagic glaucoma, with no choice but enucleation.

That enucleation was not a necessity in the case of Mrs. P. must, I think, in a great measure, be attributed to the fact that only a comparatively speaking small amount of diseased

tissue was separated at the time of the hæmorrhages. As for treatment, I believe that the repeated extraction of blood from the temporal region was of far more value than the internal medicines, in keeping the tension of the eyeball within the bounds of possible preservation. I found that the tension would sink to normal, and remain so a considerable time, after two applications of the artificial leech.—*Virginia Medical Monthly.*

RECENT EXPERIENCES IN THE TREATMENT OF DETACHED RETINA, WITH A DETAILED REPORT OF THIRTY-EIGHT CASES.[1]

BY CHARLES STEDMAN BULL, M.D., NEW YORK.

The number of cases on which the following conclusions are based is thirty-eight; of these twenty-three were men and fifteen were women.

Their ages ranged from nineteen to seventy-four. There was one case between ten and twenty years; there were six cases between twenty and thirty years; six cases between thirty and forty years; seven cases between forty and fifty years; eleven cases between fifty and sixty years; five cases between sixty and seventy years; and two cases between seventy and eighty years.

The error of refraction present was simple myopia in twenty-two cases; simple myopic astigmatism in one case; simple hypermetropia in six cases; simple hypermetropic astigmatism in one case; compound hypermetropic astigmatism in two cases; and in six cases the refraction was emmetropic.

The detachment of the retina occurred spontaneously in thirty cases, and was due to traumatism in eight cases.

The detachment of the retina was preceded by more or less extensive hæmorrhage into the retina in six cases, and by hæmorrhage into the vitreous in nine cases.

A more or less extensive *laceration* or tear in the *retina* was visible in six cases, which allowed a distinct view of the chorioid through the rent.

The intra-ocular *tension* was found normal in fifteen cases; it was subnormal in twenty cases, and increased in three cases.

The *lens* was perfectly transparent in fifteen cases, but in eight of these cases peripheral opacities appeared later. In fifteen cases there were peripheral or nuclear opacities of the lens, or both, as the time of the first examination.

The *vitreous* was generally hazy in twenty-two cases. There were floating opacities in seventeen cases and fixed membranous opacities in fourteen cases.

There was more or less marked degeneration of the *chorioid* with interstitial atrophy in twenty-two cases.

There was *iritis* at some time in the course of the disease in five cases, and acute *chorioiditis* in one case.

The *retina* subsequently became *totally detached* in nine cases, either during the treatment or at a varying period after the treament had been concluded.

The *lens* subsequently became *entirely opaque* in nineteen cases.

In one case an *extraction* of cataract was made with iridectomy before any treatment was directed toward the relief to the detachment of the retina, and with an excellent result. The wound made in the limbus healed rapidly, the coloboma remained unobstructed, the posterior capsule did not require tearing, and there was no bad effect produced on the detachment of the retina.

In one case an *iridectomy* upward had previously been done by another surgeon without producing any effect upon the detachment.

In one case the eye first affected by the disease, and which had subsequently become entirely blind, was enucleated by another surgeon preparatory to treatment of the disease in the other eye, but without producing any effect upon the condition of the second eye.

All the cases were subjected to the following treatment: The patients were placed on their backs in bed; atropine was instilled in one or both eyes, according to the necessities of

the case, and a bandage was applied to the affected eye, and this treatment was kept up for a period varying from three to eight weeks.

Pilocarpine hydrochlorate was injected hypodermically in daily doses in twenty-five cases, beginning with a minute dose and increasing it to toleration. In a number of these cases the drug produced such unpleasant or alarming symptoms that it was necessary to discontinue it. My experience has taught me that it should not be prescribed in persons suffering from functional or organic cardiac disease.

In those cases in which *pilocarpine* was not borne well, or in those in which it was contraindicated, small doses of *sodium bicarbonate* and *potassium iodide*, largely diluted, were given, the object being to induce free action of the kidneys and bowels.

Corrosive sublimate in small doses was given in the cases complicated by iritis and chorioiditis and in all cases of extensive opacities of the vitreous.

Puncture of the *eyeball* through the sclera into the subretinal space was done in nineteen cases, in every instance subconjunctivally.

Division of membranous bands in the *vitreous* and of the detached retina, thus letting the subretinal fluid out into the vitreous chamber, was done in seven cases.

As regards the results of the above *treatment*, there was a temporary improvement in the vision and in the extent of the detachment, varying from a few weeks to several years, before the vision became reduced and the detachment increased in extent, in twenty-three cases.

There was no improvement whatever in eleven cases.

There was an apparent permanent cure, with entire disappearance of the detachment and restoration of useful vision, in four cases.

There was little or no reaction following puncture of the eyeball through the sclera, or after divison of the detached retina and the membranous bands in the vitreous, in any of the cases. This fact I have already noticed in two papers on

"The Surgical Treatment of Membranous Opacities of the Vitreous" previously published, the first one in the *Transactions* of this society for 1888 and the second in the *Ophthalmic Review* for 1890.

Conclusions.—The following brief conclusions seem to be justified by the results of the treatment in the above cases:

1. The science and practice of ophthalmology have as yet discovered no better means for dealing with detachment of the retina than the old methods which have been advised and carried out for so many years—viz., rest on the back in bed, atropine, a bandage and the internal administration of some drug which may induce absorption of the subretinal fluid.

2. The continued use of pilocarpine, either hypodermically or by the mouth, may cause great prostration, even in cases in which it is apparently well borne; and the desired effect may sometimes be produced by small doses of bicarbonate of sodium and iodide of potassium largely diluted with water.

3. In all recent cases puncture of the sclera subconjunctivally may do good temporarily by letting out the subretinal fluid and allowing the retina to collapse, thus producing some improvement in the vision; but the apparent improvement is generally transient, and when membranous bands exist in the vitreous no improvement can be expected from simple puncture.

4. Division of fixed membranous opacities in the vitreous causes but little reaction, and may do positive good even without division of the detached retina, as it reduces the danger of extension of the detachment. It is positively contraindicated in cases where the vitreous opacity is vascularized, as it would certainly induce free hæmorrhage into the vitreous. It should never be done in an irritated or inflamed eye.

5. Division of the detached retina, which allows the subretinal fluid to escape into the vitreous, may always be done in a quiet eye, and causes little or no reaction. If membranous

bands are present in the vitreous, these should also be divided at the same time.

6. In most cases all these operative procedures produce but temporary improvement, and in many cases no effect whatever is gained by them.

7. There seems no good reason for any further indorsement of the method advocated by Schoeler, but every reason for rejecting it from the domain of ophthalmic surgery.—*New York Medical Journal.*